expense of these sophisticated systems (Perkin-Elmer, Centronics and Cemetron). In the future V_{O_2} monitoring will be an integral part of the ICU ventilator. During anesthesia, V_{O_2} has been monitored and studied using closed-circuit breathing circuits, in which the inflow rate of fresh oxygen is a measure of V_{O_2}.

Oxygen monitoring technology is rapidly progressing. Soon gaseous monitoring will become standard equipment on each breathing circuit. Additional improvements to and experience with transcutaneous techniques should make it possible to monitor P_{aO_2} and oxygen saturation continuously and noninvasively. Further, continuous monitoring of gas exchange, V_{O_2} and V_{CO_2} will become a standard procedure for patients needing ventilation.

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Barbiturates and Cerebral Ischemia

BARBITURATES CAN REDUCE cerebral metabolic rate, cerebral blood flow and cerebral blood volume by as much as 50 percent, and the administration of these drugs is a well-known method for controlling or reducing intracranial pressure (ICP). Recently, they have been advocated for the treatment of various forms of cerebral ischemic, hypoxic or anoxic insults (such as transient cardiac arrest, stroke, near drowning or asphyxia) or for the intraoperative "protection" (pretreatment) of patients at risk for such insults as occlusion of a carotid or cerebral artery, profound hypotension or circulatory arrest. The rationale for their use is the theory that tissue damage can be minimized if metabolic demand is reduced at a time of decreased oxygen and nutrient delivery, although other ideas have been presented (such as free radical scavenging or redistribution of regional cerebral blood flow). Unfortunately, there are discrepancies between this theory, experimental data and clinical observations.

There is laboratory evidence that pretreatment with barbiturates protects the brain in situations of *incomplete* ischemia or hypoxia (not anoxia). Barbiturate anesthesia prolongs survival in hypoxic mice, slows the metabolic deterioration of the brain (such as lactate accumulation and adenosine triphosphate depletion) during profound hypotension (blood pressure less than 40 mm of mercury) and can also reduce the size of experimental strokes produced by middle cerebral artery occlusion, at least if given before cell death has occurred (within one to two hours after occlusion). However, large doses (sufficient to produce an isoelectric EEG) are needed. Unfortunately, there are no data on humans that confirm these observations, and the clinical relevance is limited.

There is no evidence that treatment begun after reperfusion or reoxygenation is useful, except to control ICP, and there are few opportunities to "treat" patients before severe hypoxia. Similarly, the cardiovascular depressant effects make it unwise to administer large doses of barbiturates during accidental hypotension. Elective protection before planned profound hypotension (not *routine* hypotensive anesthesia) would be better achieved by hypothermia, which also can be more readily reversed, particularly if cardiopulmonary bypass is used. Barbiturates might be considered in conjunction with "emergency"-induced hypotension when there is a risk of brain damage, such as after intraoperative rupture of a cerebral aneurysm. However, the only anecdotal evidence concerns cases of elective or emergency cerebrovascular occlusion, at least when the tissue is eventually revascularized. Unfortunately, there are no post-stroke studies, principally because of the obvious concern with cardiovascular and respiratory depression in elderly patients. Therefore, with the exception of the hypothetical uses just mentioned, it is difficult to support the use of these drugs. Barbiturates should not be used routinely as an adjunct to carotid endarterectomy, because most patients tolerate transient cross-clamping without problems. If clamping produces ischemia (EEG depression) that cannot be reversed by raising blood pressure or by shunt placement, such use might be considered.

The most controversial use of these drugs is after resuscitation from a cardiac arrest. Recent debate was prompted by a single primate study, which has been questioned on both procedural and theoretical grounds. Other studies have not confirmed the efficacy of barbiturates given before or after a period of circulatory arrest, in contrast to the proven usefulness of hypothermia before

the time of the arrest. More importantly, uncontrolled trials have shown no benefit, and results from a preliminary nonrandomized study of thiopental given after resuscitation have shown no improvements in mortality or neurological morbidity. Therefore, there is *no* evidence supporting the "blanket" administration of these drugs to patients following cardiac arrest, although they may prove useful in treating postanoxic seizures in some persons. There is, however, some circumstantial evidence supporting an aggressive approach to these patients, and careful respiratory and cardiovascular support may be of value.

In spite of intriguing laboratory evidence, the clinical role of barbiturates in brain protection remains unproved, except for control of ICP (which probably has nothing to do with "protection" per se). In highly select intraoperative situations, their use can be justified—if used with great caution. However, there is no role for these drugs in treating patients following cardiac arrest.

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